MASKED CARDIAC INFARCTION

Eight Case Histories

By BRANFORD MORGAN, M.D., F.R.C.P. (Lond.), F.R.C.P. (Edin.)

Norwich

Since Herrick in 1912 described the effects of sudden occlusion of a coronary artery, an extensive literature has so clearly portrayed the clinical picture that infarction of the myocardium but rarely passes unrecognized. Equally well known is its simulation of other acute thoracic or abdominal catastrophes, and its liability to be followed by thrombotic and embolic complications.

Apart from a case quoted by Parkinson and Bedford (1928) of cardiac infarction which presented owing to gangrene of the leg due to embolism of the femoral artery, little or no attention appears to have been drawn to the possibility that such thrombo-embolic complications may be the presenting symptoms of cardiac infarction; or that cardiac infarction may be masked by severe coincident biochemical disorders such as diabetes or uraemia. It is well known, and was indeed stressed by Gallavardin in 1921, that patients may die from infarction of the heart without suffering pain, and recent papers have emphasized the frequency of infarction with minimal discomfort. If treatment by anti-coagulant substances is to be instituted early recognition of myocardial infarction is of prime importance. The following illustrative cases are briefly described:

Case 1. Aphasia and right hemiplegia. A man, aged 42, was found unconscious in bed with a right hemiplegia. With return of consciousness on the following day he was found to be aphasic. His heart was clinically normal, the rate 80, rhythm regular, blood pressure 120/80. Owing to the aphasia no history was available to assist in elucidating the cause of the sudden hemiplegia. His wife, when visiting two days after admission, told us that six days previous to the cerebral episode the patient had complained of severe pain in the chest lasting a few hours, thought to be indigestion, and for which he did not seek medical

advice. An E.C.G. showed a recent antero-septal infarct. The hemiplegia was therefore probably due to an embolism from an intramural thrombus.

Case 2. Hemiplegia. A man, aged 65, said to have been previously in good health, whilst talking to his wife was violently sick and lost the power in his left arm and left leg. The day following his doctor noted auricular fibrillation lasting for 24 hours. On examination 48 hours after the onset of hemiplegia the heart was regular, rate 100, blood pressure 170/120. An E.C.G. showed a recent antero-septal infarct. After pressure, the patient admitted to typical effort angina for some time previously of which he had taken little notice and for which he had not consulted his doctor. The hemiplegia was presumed to be due to a cerebral embolism from a transient auricular fibrillation.

Case 3. Multiple emboli in a diabetic. A male diabetic, aged 62, years, who had been treated by his doctor with insulin for the last ten years, was sent into the ophthalmic department of a general hospital. He was extremely ill and stated that his vision had suddenly become blurred and that he had noticed for the last two days increasing weakness of his legs and lower abdominal pain. These symptoms had been ascribed by his doctor to diabetes. When seen by a physician, both legs were completely ischaemic. There was no femoral pulse in either leg. There was a left hemianopia with no fundal changes. The urine was loaded with sugar and ketones, blood sugar was 340 mg. per cent. The heart sounds were normal, rate 110 blood pressure 135/80. He died within an hour of admission. At autopsy a mural thrombosis, the size of a plum, was found in the cavity of the left ventricle over the site of the recent infarct. Emboli were found blocking the upper ends of both femoral arteries; 18 in. of the lower ileum and the occipital pole of the right cerebral hemisphere were

infarcted. It was subsequently ascertained that three weeks previous to admission he had had a severe gripping pain across the lower chest, spreading to the abdomen.

Case 4. *Simulating pneumonia.* A woman, 57 years of age, was sent into hospital as a case of pneumonia, resistant to sulphonamides and antibiotics. A week previously she had been seized with pain in the left lower chest of pleuritic type with productive cough, breathlessness and pyrexia. On Admission, temperature was 100° , rhonchi were audible throughout both lungs and there were moist sounds at the base of the left lung. X-rays showed no significant abnormality. The heart was clinically normal, rate 90, blood pressure 170/105. Following attacks of nocturnal dyspnoea an E.C.G. was taken and showed a recent posterior cardiac infarction. On inquiry she admitted to having had an attack of lower trans-sternal pain seven days before the onset of the chest pain for which she had summoned her doctor. It was considered that the left pleuritic pain was due to a pulmonary infarct caused by an embolus from a mural or leg vein thrombosis.

Case 5. *Pleural effusion.* A man, aged 66, was admitted to hospital with a history that one week previously he had experienced gradually increasing pain of pleuritic type below the left costal margin with progressive effort dyspnoea. He was found to have a moderate sized pleural effusion with fever ranging from 100° to 102° which continued for 14 days after admission. The white cell count was 10,000, heart rate 70, blood pressure 140/80. The pleural fluid was lemon-yellow, sterile, 40 per cent. polymorphs, 60 per cent. lymphocytes. Owing to transient irregularity of the heart an E.C.G. was taken which showed a recent antero-septal infarct. On direct questioning he admitted to praecordial pain on effort for some weeks before admission. The pleural effusion was considered to be the result of a pulmonary infarct from a mural or leg vein thrombosis.

Case 6. *Simulating 'status asthmaticus.'* A female, aged 64, was sent into hospital as a case of status asthmaticus of four days duration. On admission she did appear to be in status asthmaticus, severely dyspnoeic, slightly cyanotic, with no increased pressure in the neck veins and no oedema. She had a slight non-productive cough. She denied pain, past or present. Her heart sounds were normal but blurred by added lung sounds. The heart rate was 110, rhythm regular, blood pressure 150/80. Her 'asthma' was unrelieved

by routine measures. X-rays on the day following admission showed a little fluid at both lung bases. An E.C.G. showed recent anterior cardiac infarction.

Case 7. *Simulating diabetic pre-coma.* Five days before admission to hospital a man, aged 71, complained to his doctor of upper abdominal pain which had kept him awake the previous night. Two days after he collapsed in the back yard. He walked indoors but owing to slight mental confusion and restlessness his relatives called in the doctor. To the latter he denied pain in his chest or elsewhere. His urine being loaded with sugar and ketones he was sent to hospital as a diabetic pre-coma. His blood sugar was 320 mg. per cent., the abdomen was normal, heart clinically normal, rate 80, blood pressure 185/110, there were no signs of focal intracranial disease. An E.C.G. showed a posterior infarction, no doubt the cause of the abdominal pain, subsequent collapse and mental confusion. It was presumed that the diabetes was present prior to, but was aggravated by, the attack of cardiac infarction.

Case 8. *Simulating diabetic or uraemic coma.* A chemist, aged 76, an established but only moderately controlled diabetic, after a days' shooting, experienced pain to the left of the mid-sternum which lasted throughout the night. The following day he was free from pain, but developed constant vomiting for which he was sent to hospital as a diabetic or uraemic coma. On admission he was drowsy, cold and blue. He had continuous vomiting and hiccough. There was mild glycosuria with moderate ketosis. The blood sugar was 540 mg. per cent., blood urea 90 mg. per cent. The heart rate was 36, blood pressure 120/0. E.C.G. showed a recent posterior cardiac infarct with complete heart block.

The occurrence of infarction had upset his diabetic balance and presumably an associated fall in blood pressure had resulted in diminished renal blood flow and consequent rise in the blood urea level.

Summary

Attention has been drawn to the danger of missing an underlying cardiac infarction if the patient presents on account of a thrombo-embolic complication or a grave metabolic disorder.

BIBLIOGRAPHY

- PARKINSON and BEDFORD (1928), *Lancet*, 4, 11.
GALLAVARDIN (1921), *Jour. Med. de Lyon*, 1, 138.